(PAM) 616-002 5

TUBERCULOSIS

AS AN ENDEMIC DISEASE.

INAUGURAL ADDRESS.

Read before the EPIDEMIOLOGICAL SOCIETY OF LONDON,
SESSION 1892-93.

BY

JOSEPH FRANK PAYNE, M.D., F.R.C.P., PRESIDENT.

LONDON:
PRINTED BY CHAS. J. CLARK, 4, LINCOLN'S INN FIELDS, W.C.
1893.

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The Epidemiological Society of London.

INAUGURAL ADDRESS OF SESSION 1892–93.

ON TUBERCULOSIS AS AN ENDEMIC DISEASE.

BY JOSEPH FRANK PAYNE, M.D., F.R.C.P., PRESIDENT.

(*Read: November* 16*th*, 1892.)

GENTLEMEN,—In taking the chair of this distinguished Society, I must commence by thanking you for the honour you have done me in electing me to this office, and expressing my sense of the great responsibility of following the numerous eminent men who have preceded me in this chair.

I have felt some difficulty in selecting any topic for my remarks to-night, which I could hope to present in a manner at all worthy of your hearing; but I hope that the subject of tubercular disease will not be regarded as foreign to the aim and scope of this Society. Our aim, as I find it defined in earlier addresses from this chair, has always been the investigation of the origin, propagation, mitigation, and treatment of epidemic diseases. The word epidemic has not, however, been understood to exclude the consideration of diseases which are rather endemic than epidemic; that is, looked at in relation to their distribution in space, rather than to their distribution in time. Moreover, phthisis, or tubercular disease in general, has been recently made the subject of several papers read before the Society, and we may assume that the study of this disease is now, whatever it may have been formerly, regarded as coming within the scope of the science of Epidemiology, taken in a wide sense.

If we enquire into the presumable reasons for this inclusion of tubercular disease in our province, we must, I think, assign it partly to a change which has recently taken place in our manner of regarding this disease.

The starting point of our modern conception of tubercle was unconnected with and anterior to any doctrine of its being caused by a micro-organism. Villemin, in 1865, or earlier, had the happy thought of trying whether tubercular diseases could be conveyed by inoculation of tubercular products, like vaccinia and other inoculable diseases. His experiments gave, in the case of rodents, a positive result; and from that time, supposing Villemin's conclusions to be, as they were, confirmed by others, tubercular disease had to be transferred to the great class of specific diseases; that is, of those which are caused by some definite material virus, and not by various kinds of injuries, or by quantitative variations in the more general causes of disease, depending on nutrition, climate, and the like influences.

I need not recount the steps by which the undoubtedly serious objections brought against Villemin's theory by Wilson Fox, Burdon Sanderson, Cohnheim, and others were finally got rid of (as confessed by all these investigators themselves); till in 1881 Cohnheim was able clearly to formulate the theory that tubercle was due to a specific communicable cause, even though this cause was still unknown.

Even had this material cause never been actually discovered, the specific nature of tubercle would rest on the same footing as that of small-pox, scarlatina, and other exanthemata, with respect to which the same uncertainty as to the precise nature of the specific virus still remains.

Koch's brilliant discovery in 1882, identifying this unknown cause with a definite bacillus, came when the times were ripe.

The history of discovery in this subject has been the same as in the investigation of many obscure natural phenomena. The first step is to frame the hypothesis of a cause adequate to account for the phenomena: operating at the right time and in the right place. Further, it must be shown that no other cause is capable of producing the same effects, since it is not impossible *à priori* that the phenomena may be produced by more causes than one. But this will not be enough, since we can evidently invest a hypothetical cause with any properties we please. It must be shown that the supposed adequate cause really exists, and operates at the time and place supposed. The results of Villemin and his followers clearly showed what kind of cause was necessary to explain the occurrence of tubercle, and that no other of the causes assigned, such as inflammation, cachexia, etc., was adequate. It remained for Koch to show that such a cause, perfectly adequate to explain the production of tubercle, actually existed, and was in operation at the time and place necessary to account for the phenomena of tubercular disease.

By these discoveries it has been clearly established that *tuberculosis* (and this modern name is in itself significant) belongs to the class of diseases with which the Epidemiological Society chiefly concerns itself.

It is true that tuberculosis differs from many of the diseases called specific in certain particulars—in its insidious onset and long duration, in having no self-determined limit of existence, and hence in not conferring (as is probable) any immunity against itself. Moreover, its mode of communication is obscure, and hence, some think, is not well expressed by the words infectious and contagious, which carry with them certain vague connotations. Nevertheless, it cannot be doubted that it is, in the strictest sense, communicable, for the inoculation of tubercle to animals is as certain as the inoculation, for instance, of vaccinia. Again, the prevalence of tuberculosis in man is much affected by external circumstances, by climate and conditions of soil, by density of population, by inherited constitution, and other influences. Still there is no one of these conditions which does not affect the prevalence and spread of some one or more of the diseases called specific. These facts do not at all disprove its causation by a definite material, living, and communicable virus, that is, by the bacillus of Koch, the only known cause which in any way accounts for its phenomena.

ENDEMIC EXISTENCE OF THE TUBERCULAR DISEASE.

The problem which I propose specially to consider is, what are the conditions which maintain the continued existence of tuberculosis as an endemic disease in any country or population? If the disease inevitably depends upon a certain organism, the tubercle bacillus, then the problem resolves itself into two: What are the conditions which maintain the continued existence of the tubercle bacillus? and, What are the conditions, if any, which would make the population proof against attack, *i.e.*, give immunity against the bacillus, even supposing it to be widely prevalent?

The second of these problems might be of great practical importance if the bacillus were regarded as ineradicable, or, at least, little affected by preventive measures. Whether its solution will ever be found we cannot say. I do not in the least undervalue the importance of trying to find its solution. But, on the principle of one thing at a time, I shall not further refer to it, but confine myself to considering the causes of the prevalence or continued existence of the tubercle bacillus.

Continued Existence of the Tubercle Bacillus.

The continued existence of any living organism in any country will obviously depend upon its finding an appropriate habitat and abundant food, and also on the presence or absence of conditions militating against its existence.

As regards the tubercle bacillus, there is no doubt that its chief habitat is the human body, where it finds, as a parasite, its appropriate pabulum; and the conditions limiting its existence are only the conditions of health or the reverse which affect its vitality there. But it is also a very important question whether it can really live, that is, grow, outside the human body, in a saprophytic or ectogenic existence; or, again, whether it can at all events maintain its vitality, without growth, in such conditions, so as to be ready for development when introduced into the body again.

Koch, finding that he was unable to cultivate the bacillus except at temperatures between 82° and 105° F., and that the best temperature was 99.50° F.—that is, the temperature of internal parts of the human body—concluded that it was a true parasite, and incapable of growing in external nature, at all events in our climate. This conclusion is confirmed in the strongest manner by most bacteriologists. I find only one contrary observation, that of Sir Hugh Beevor, who, using the improved methods of cultivation introduced by M.M. Roux and Nocard, found that the bacillus would grow at the temperature of an ordinary room in winter, which was rarely above 60° F., and often much lower, though the growth below 99° F. was decidedly slow.*

These results seem to require confirmation, as I find they are not confirmed by the experience of most bacteriologists, but, if confirmed, they would show that the bacillus is capable of an ectogenic existence, and is not a true or obligatory parasite, but an occasional, or, as it is said, "facultative" one. Dr. Andrew and others support the same view on theoretical grounds. It is also evident that, even according to Koch's conclusions, the temperature of air and soil in hot climates would not be unsuitable for the bacillus, provided sufficient moisture were present. On the other hand, all bacteriologists agree that the bacillus is extremely sensitive, or, so to speak, difficult to please in the matter of artificial cultivation-media, very few of the customary media suiting it, so that its artificial cultivation is one of the most difficult problems in practical bacteriology.

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* *Trans. Path. Soc.*, London, 1891, vol. xlii, p. 344.

As regards its pabulum, it is found that moist albuminous materials, simple or peptonised, are the one essential constituent. Further, it has been found by Nocard and Roux that a product of the decomposition of fat, glycerine, accelerates the growth in a remarkable manner.*

It should be added that moisture, necessary to the growth of all bacteria, is preeminently so in the case of the tubercle bacillus, since the slightest drying of the surface of the cultivation-medium stops its growth, though without destroying its vitality. The bacillus is also aerobic, that is, needs a certain amount of oxygen for its growth. Now moist albuminous materials, mixed with products of fatty decomposition, are certainly present in or about the abodes of men and animals (though not in the open soil); and might conceivably, if at a proper temperature, form a suitable nidus for the growth of the bacillus. Nevertheless, we must conclude that the possibility of an ectogenic or non-parasitic growth of the bacillus is not certainly proved, and is, on the whole, in our climate, improbable. The question of such possibility in hot climates must remain for the present an open one. And it is worthy of note that a climate at once hot and moist seems, generally speaking, the most favourable for the spread of tubercular phthisis, as shown, for instance, by the higher mortality from phthisis in the British army in the West Indies than in Canada. Further, the belief in the contagiosity of phthisis (which would certainly be favoured by the conditions supposed) has always been most strongly held, and is so still, in comparatively hot countries such as southern Europe.

Independent of the possibility of growth and multiplication in external nature, there is an important possibility in the case of the bacillus, viz., whether it can maintain its *vitality* in conditions not admitting of actual growth, so as to be capable of growing again in more favourable circumstances. Most bacteria, like other organisms, animal or vegetable, have the power of concentrating their vitality into special products, like seeds, capable of resisting adverse influences which would be fatal to the growing organisms. These products, viz.,spores, are well known in many bacteria; but with regard to the tubercle bacillus there is considerable doubt as to whether the real spore has been found. The well-known beaded condition of the bacillus as seen in phthisical sputum is often spoken of as spore formation. But I must confess to an individual scepticism on this point. It is not proved that either the portions stained by ordinary reagents, or those unstained, are true spores. It is thought that the beaded or supposed spores containing bacilli are more resistant and tenacious of life. But, in fact, it is almost impossible to separate the apparently sporeless from the beaded forms, and Baumgarten has arrived at the conclusion that the difference in point of vitality observed between these forms in some species of bacteria does not hold in the case of the tubercle bacillus.*

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* Nocard and Roux, *Annales de l'Institut Pasteur*, 1887, No. 1, p. 19; Crookshank, *Trans. Path. Soc.*, 1891, vol. xlii, p. 330.

However, spores or no spores, the bacilli have great powers of resistance to injurious influences—to dryness, to heat, to cold, to contact with putrefying substances, and to antiseptics. After being dried up for several months at ordinary temperatures they are still capable of growing and multiplying if introduced into an animal body, but several circumstances modify the possibility of revival. At a temperature of 86° to 104° the vitality declines much more rapidly than at ordinary air temperatures. But, in any case, after nine to ten months at the outside (some say after only two or three), their vitality is completely lost. By contact with putrescent fluids the bacilli are killed much more rapidly, even while their form remains unchanged, and they are still capable of being stained. Only in a very extreme case was putrefying tubercular sputum found to be capable of conveying disease after forty-three days. On the other hand, very low temperatures do not destroy, but only check, the vitality of the bacilli which remain capable of revival. I do not here speak of artificially high temperatures, or of antiseptics, since I am considering only conditions which may occur in nature.

The influence of light has been held to be important. Koch stated at the Berlin Medical Congress of 1890 that exposure of the bacilli to direct sunlight for a few days destroys their vitality, but the precise experiments on which this statement was founded have not, I believe, been published.*

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* Baumgarten, *Lehrbuch der pathologischen Mykologie*, ii, 553. This work contains numerous references on the subject of the vitality of the tubercle bacillus.

There can be no doubt that the conditions necessary for the preservation of the bacillus in a condition of quiescent vitality are present in climates such as ours. The question may arise, however, whether they are more likely to occur in the soil or in inhabited dwellings.

With regard to the open soil, whatever may be experimental possibilities, there is little concrete evidence for believing that it can be the nidus of the tubercle bacillus; that is that the tubercular virus is ever a soil-poison. For tuberculosis has never been known to be acquired by passing through a particular country, or sleeping in the open air, as in the case of ague; there is no evidence of its being communicated by accidental or intentional inoculation with earth, like tetanus; or by contaminated drinking-water like typhoid or cholera. Broadly speaking, its prevalence rises or falls with human, not with miasmatic conditions.

DAMPNESS OF SOIL IN RELATION TO PHTHISIS.

There is, however, one important condition of the soil, the connection of which with pulmonary consumption has been worked out by the classical researches of Sir George Buchanan, one of our former Presidents. He showed, by the law of concomitant variations (to use Mill's formula), that in certain portions of England the mortality from phthisis stood in a direct relation to the moisture of the subsoil; this being determined by the perviousness of the soil and the facilities for natural drainage. Further, he proved that a remarkable improvement in the death-rate from phthisis followed, in many English towns, the carrying out of extensive drainage-works, which had for their primary object the carrying off of sewage, but also led to drying of the subsoil. This result might be partly attributed to general sanitary improvements, especially as the lowering of the death-rate from typhoid fever was still more striking; but it is extremely significant that *the death-rate from other pulmonary diseases did not diminish in the same ratio*, in most cases did not diminish at all, and in some instances was even increased, during the period to which these observations refer. The independent researches of Dr. Bowditch in America strikingly confirm these results.

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* These statements as to the injurious action of sunlight on the growth of the bacillus have not been confirmed by other investigators. Dr. Ransome (*Proceedings Royal Society*, xlix) found that exposure of tubercular sputum to light, *in conditions otherwise insanitary*, did not destroy its virulence, as tested by inoculation, but exposure to fresh air and light on a dry soil had that effect.

Sawizky found that dried tubercular sputum lost its virulence in two months and a half, but the change took place in the same time in darkness as when exposed to direct sunlight.

A remarkable parallel to Sir G. Buchanan's results is presented by those recorded in a certain district of Germany by Dr. Finkelnburg, as presented to the *Congress für innere Medizin* at Wiesbaden, 1889 (*Berliner klinische Wochenschrift*, 1889, p. 434). The district of Meppen, in North Germany is a purely agricultural one, and among the most scantily populated in Germany. It is a moorland country, abounding in bogs, where the water collects. In this district the mortality from phthisis *in the female sex* is higher than in any other district. The same high rate of female phthisical mortality is, according to Dr. Finkelnburg, found in the adjacent parts of Holland and of Oldenburg; though in these parts the social conditions are different, and the only factor in common is the character of the soil. This excess of mortality from phthisis in the female sex has other bearings, which I shall refer to again.

On the other hand, it must be said there are several observations showing that in certain towns drainage has not diminished the death-rate from phthisis; and in some instances even an increased rate has followed such operations. And in certain districts of Sussex the diminution in mortality from phthisis, which was traced during the time of Sir G. Buchanan's observations, has been found by Dr. Kelly to be altered, or even inverted, in subsequent periods.

The laws of malaria, too, seem in some degree to contradict Sir G. Buchanan's conclusions. It has often been stated that malaria, or ague, which in our climate is so closely related to dampness of the soil, stands in an inverse ratio to phthisis as regards its prevalence in particular districts. This is a controverted point, on which I cannot enter; but one broad fact must not be forgotten, that malarious districts are, as a rule, thinly populated, while consumption is directly related to density of population; and hence, when certain districts are cleared of malaria, and become more thickly inhabited, phthisis is likely to assume a larger place in the statistics of mortality. Notwithstanding these objections, we cannot doubt that dampness of soil is, on the whole, a favouring condition for the occurrence of phthisis. Whether it is so for other tubercular diseases, I am unable to say.

However, I wish to point out, as showing that tuberculosis cannot be strictly a soil-disease, that this influence is by no means a preponderating one, at least to the extent that it is in ague, in cholera, in typhoid, and some other maladies.

Ague has been notoriously almost exterminated in our own country during the last century, or, at the outside, two centuries; and there is nothing which can account for so universal a disappearance of the disease but the thorough drainage of the soil, which is regarded as an essential in modern agriculture.

But, with regard to phthisis, this is by no means the case. Nothing approaching to extinction of tubercular disease has been seen in any part of the country. The subsoil of London, for instance, is now remarkably dry, and must be getting drier every year, owing to the covering so large a proportion of the area with houses, and the remainder with pavements, almost or quite as impervious, so that only a small portion of the rainfall penetrates the soil at all. Except in our parks, gardens, unpaved yards, and the like, it is immediately carried off into the drains: a state of things which, over so large an area, can, I suppose, hardly be paralleled in the world, and which, even in our rainy climate, must inevitably tend, in the long run, to drying up of the subsoil, even to a considerable depth. Notwithstanding this, tubercular diseases, though they are decidedly diminishing, still flourish among us. On the whole, we cannot conclude that the condition of the soil is the main factor influencing the prevalence of tuberculosis, as would be the case were the virus of this disease a soil-poison.

CONNECTION OF TUBERCULAR DISEASE WITH DWELLINGS.

It might be suggested that the influence of damp soil in favouring consumption is exerted by making the *houses* damp, and therefore unhealthy. I do not know any observations bearing on this point, beyond the general impression that "insanitary" dwellings foster tubercular disease. Of late years this question has assumed a somewhat different shape. It has been suggested, and in many instances proved, that a special prevalence of phthisis attaches to particular dwellings, so that special localities in a town, special streets, special houses in a street, even special rooms in a house, have appeared to be nurseries of tubercular disease. This fact, I need hardly say, has been often observed in the case of other specific diseases. It was noticed in London, in Vienna, and other cities during successive invasions of the plague, in the seventeenth century, that the disease broke out in the same houses where it had prevailed in previous epidemics. With regard to typhus, diphtheria, and other infectious diseases, the fact is too well known to need dwelling upon. The question is, whether the same law holds with regard to tubercular diseases; that is, whether the infection, if it be called so, clings to particular houses, and not merely to houses having the same sanitary defects. That certain sanitary defects favour the occurrence of tuberculosis has, of course, been often pointed out. It has been shown unmistakably that imperfect ventilation and overcrowding of dwelling-houses or workshops greatly favour the prevalence of consumption. The often-told story of the high mortality from consumption in the English army, irrespective of climate, and the immense improvement which has resulted from improvements in barracks, is familiar to all. The excessive prevalence of the disease in prisons and asylums in former times, and the comparative exemption of such institutions at the present day, tell the same tale. Many histories of convents tell it still more clearly. From these facts was drawn a conclusion which has great influence on medical opinion as to the causation of phthisis; viz., that the breathing of air deteriorated by organic impurity, or, as some put it, of rebreathed air (to use the words of Sir James Clarke in 1835), is one of the most powerful favouring causes of the disease. The researches of Farr, McCormac, Parkes, Welch, and, more recently, of Dr. Arthur Ransome, Dr. Barry, and Mr. Gordon Smith, and others, have fully confirmed the earlier conclusions.

Before, however, committing ourselves to an unqualified faith in this theory, let us listen for a minute to a voice from the Antipodes. Mr. T. N. Fitzgerald, President of the Intercolonial Medical Congress, held at Melbourne, 1889, stated the results of his large practical experience as follows :—

"We have been long taught, rightly or wrongly, that scrofula and tubercle are affected by squalor and dirt; that phthisis is favoured by close vitiated atmosphere and cold night air. These causes are present but to a slight extent with us, yet occasionally we find scrofulous impregnations excessively virulent; while phthisis is as common with us as it is in England; and decidedly more so than in Canada and Scotland."

* * * * *

"In my early days, it was impressed upon us that consumption chiefly arose from defective ventilation, and the inhalation of irritant particles. Then came the catarrhal or pneumonic origins, neglected colds, or so forth; and now we find that neither nor all of these reasons will suffice to account for the large bills of mortality from phthisis in this land.

"In my opinion, next to its hereditary inception, the greater part of phthisis will be found to be associated with defective drainage; and this, I believe, will be proved by bacteriology."*

There can be no doubt that the social status of the labouring class in the colonies is superior to that of the same class in this country; and that most of the conditions so often referred to are not widely prevalent there. Comfort is far more generally diffused, and the supply of food is generally abundant; while the milder climate does not encourage overcrowding in the same way as our humid skies and treacherous weather. Nevertheless, the death-rate from phthisis in Melbourne in 1885 was 2391.6 per 1,000,000 living; while in London, in 1887, it was only 1858 per million. The only circumstance in which Melbourne is comparable to the English cities half a century ago is the extremely defective state of the drainage.

It is possible that we exaggerate the force of these insanitary conditions; and it must not be forgotten that while the term "overcrowding" is modern, the fact which it denotes is ancient, not to say prehistoric. In cold countries it has always been the custom for men, as well as social animals, to huddle together at night for the sake of warmth. It is only latter-day ideas which make this a sanitary crime; while ventilation is distinctly a modern virtue. It seems to me difficult to regard these influences as more than favouring circumstances, aiding the operation of a cause more potent and more subtle. Admitting, however, the operation of such influences in this limited sense, their significance may be interpreted in two different ways.

Either these influences may act by deterioration of the general health, or by facilitating the conveyance of infection. The first mode of action is clearly seen in the influence which the same conditions have, for instance, in producing anæmia, which, apart from certain cases of developmental change, I believe to be produced in the labouring population of London far more directly by working in crowded, ill-ventilated rooms than by any other cause.

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* Presidential Address to Intercolon. Med. Congress. Report, 1889, p. 13.

On the other hand, it may be a question of direct infection. No proof is needed that contagion increases in an intensified ratio with crowding in a limited space; it seems to be governed by a law like that of the inverse squares in the case of gravitation; and might probably receive a similar mathematical expression. Inductively, also, experience supplies abundant proof of the intensification of any contagion when confined within narrow limits.

This will be the case whether the infection is derived from an infected individual, or from any source of external infection.

It may be a question which of these explanations best accounts for the connection of tubercular disease with insanitary conditions of houses.

For my own part, I decidedly incline to the latter explanation: that the insanitary conditions of dwelling-houses, so far as they favour the spread and malignancy of tubercular disease, do so by fostering the growth of a specific contagion already present there; and the mere production of a cachectic state in the inhabitants, though it may be a contributory cause, is not sufficient to explain the facts.

In support of this conclusion I will review the evidence as to the existence of a specific infective agency in dwellings which have been observed to foster tubercular disease.

From my own experience I can say little about these relations. But some years ago I observed one case which impressed the possibility of such a connection very strongly upon my mind. A tradesman from a country town came to consult me, who had well-marked signs of phthisis. He was a well-built, robust man, in good circumstances, and was himself puzzled to understand why he should have become consumptive, considering his generally good constitution, and an entire absence of any family predisposition. He said he had been ill after moving from another town to that in which he then lived; and he was much impressed by the fact that two previous occupants of the house, quite unconnected with him by family, had died of consumption. There was one suspicious circumstance connected with the house, namely, that a well was sunk in the basement. This, of course, was a factor of dampness. Whether, therefore, the determining cause of the phthisis was dampness, or whether it was an infection left by the previous occupants, I leave undetermined. There is no doubt as to the occurrence of sporadic instances like the solitary one which I have been able to contribute from my own experience. Such instances may easily be multiplied; and are only liable to the objection that in each case there may be mere coincidence—an objection which disappears when the numbers are considerable. Another group of facts is derived from the valuable researches of Dr. Arthur Ransome, detailed in a paper on "Some Evidences respecting Tubercular Infected Areas", contributed to the *Transactions of the Epidemiological Society* (vol. vi, N. S., p. 124).

Dr. Ransome says that his attention was first called to the subject by some striking instances of whole families *living in the same house* being carried off in succession by the disease; in one family of six members, the mother, an old woman, was the only survivor. In another, the only one, out of four or five, to escape was a daughter, who was in service elsewhere. He also quotes cases from the *Collective Investigation Record* to the same effect.

Three of these I will briefly refer to (Nos. 188, 194, 196). In each case a member of a previously healthy family came home ill of consumption, and several—*i.e.*, the majority—of the members of each family, though previously healthy, became affected with the disease and died of it within from two to six years.

Such cases, though many more might be quoted, are liable to the objection that family predisposition was the cause of the complaint. Greater weight, therefore, attaches to another case quoted in the *London Medical Record*, July 1884, on the authority of Dr. Kempf, of Louisville, U.S., in which phthisis appeared to be introduced into a convent, said to be well situated and ventilated, with the result that, within four months, nine sisters, regarded as exceptionally healthy, became consumptive, and four died in the course of the year. After complete isolation of the sick the epidemic was stopped. Dr. Ransome justly expresses some doubt as to the sanitary condition of the convent; but still, the fact of the introduction of the disease into a building, and its eradication (by preventive measures), is not weakened thereby. Dr. Ransome's account of the prevalence of phthisis in certain streets of badly-built houses, deficient in supply of fresh air, is most striking. But what I particularly select from his statistics is the number of instances in which two or more deaths from phthisis occurred in the same house. "Altogether", he says, "there were twenty-one such coincidences", and fifteen of them (thirty deaths in all) occurred in narrow streets, culs-de-sac, or small courts. This was in a district in Ancoats, Manchester, in a period of five years.

In a district in Greengate (Salford) eight deaths were in four houses, or four coincidences. In another district, out of forty cases in six years, ten were in five houses. The distribution of these deaths as to families is not given.

In his Milroy Lectures "On the Causes and Prevention of Phthisis" (p. 65), Dr. Ransome quotes also a personal communication from Dr. Niven of Oldham, showing that out of 3,001 deaths from tuberculosis during eleven years, in 302 instances there were two or more in a house. Dr. Niven calculates mathematically that the chance of any house being twice affected (independent of infection), only 68 houses would have suffered, whereas 274 were thus attacked, and that whilst not more than 7.6 houses should have suffered thrice, the actual number so attacked was 24.

Dr. Ransome also gives the following results arrived at by Dr. Flick of Philadelphia (whose original memoir I have not been able to meet with), from a minute topographical study of phthisis in that city during 25 years:—

(1) A house which has had one case of consumption will probably have another within five years, and may have a large number of cases in close succession.

(2) When a case of consumption occurs in a house, approximate houses are considerably exposed to contagion.

(3) Houses in *localities* where endemic after endemic has existed, have nevertheless escaped the disease.

(4) Tuberculosis of different kinds occurs in the same localities, and often on the same lots as consumption.

(5) During the twenty-five years, scarcely 20 per cent. of the houses of the ward were so affected.

He ascribes these results to contagion in the houses themselves.

Another still more important class of cases is that in which a house or room which has been occupied by a patient with phthisis gives rise to tubercular disease in subsequent occupants.

Dr. Janeway of New York (*Archives of Medicine,* quoted in *Brit. Med. Journal,* 1883, i, 377) gives the following case:—

"A gentleman died of phthisis after two years' illness, in February 1882. His sister and her husband occupied his room after his death. The brother-in-law began to fail five weeks afterwards, and in May was the subject of acute phthisis."

Dr. Burney Yeo mentions, among others, the following (*Brit. Med. Journal*, 1885, i, 774):—

"A fine young Scotch girl, of a healthy, long-lived family, was afflicted with phthisis. On enquiry it was found that some few months before she had been given a bedroom which had just been quitted by a maidservant with phthisis."

Dr. Niven (*Public Health*, ii, 206) gives a very similar case:—

"A married woman from Oldham went with two children (one a lad ten years old) to Southport, to join her husband, in September 1887. The husband, wife, and the two children all slept in one front room. In the same house was a woman suffering from consumption, said to have been contracted from her husband, who had previously died of the same disease. As the weather got colder, this consumptive woman asked them to change rooms with her, as her bedroom was colder than theirs. This was done. The Oldham woman used to complain of a peculiar odour in the room, which does not seem to have had any special cleansing or disinfection. In January 1888, the boy, ten years old, died of 'inflammation of the brain'; he had been ill a month, and from the history, his fatal malady was almost certainly tubercular meningitis. The Oldham woman became pregnant in Southport, but removed shortly afterwards to Oldham, and was there confined in June 1888; but before the birth of the baby she lost flesh, and showed the first symptoms of phthisis. The baby died in June 1889, of tabes mesenterica, and she died in October, of phthisis."

Another illustration is quoted by Dr. Heron (*Evidences of the Communicability of Consumption*, 1890, Appendix, p. 153) from Kruche (*Deutsche Med. Zeitung*, 1882, p. 445):—

"A young robust lawyer spent some time at a health-resort; and some weeks after his return home showed symptoms of a rapidly fatal attack of consumption. It was found on inquiry that in the place referred to he had occupied a room and bed just vacated by a consumptive, who had suffered from suppurating sores."

The most striking example of this mode of communication which I have met with is related by F. Engelmann (*Berlin. klin. Wochenschrift*, 1889, Jan. 7, p. 6), and relates to a particular dwelling (or "flat") in a large house occupied by the families of several glass-blowers employed in a certain factory. This occupation is one known to produce remarkable liability to phthisis; and Engelmann believes that 75 to 80 per cent. of the workmen in this particular place die of it. It is well known that this frequently has been ascribed, among other causes, to direct infection from mouth to mouth by the blow-pipe. However, that would not explain the special incidences here described. The house was built about 1865, and was generally in tolerable sanitary condition. The particular dwelling in question was badly ventilated and lighted. It was occupied from 1865 to 1873 by three families in succession who were all healthy. In 1874 a family named Nestle entered, in which the mother was already consumptive, and had lost a son from the same disease. She died there from hæmoptysis, and shortly afterwards the family left, having lived there for one year only. The next occupants were a family (Gotz) of seven persons, all in good health, though there was a history of chest disease in the mother's family. After one year's residence in this dwelling they left, and at a later time it was found that the father, mother, and one son had died of phthisis, and a fine boy of chronic peritonitis. The third family (Gunnel) occupying the dwelling were healthy on arrival, and had no hereditary taint. A child born in the house died of meningitis; another child died of "marasmus"; another boy acquired hip-joint disease. Later on, the father died of phthisis, and another child of meningitis; the mother became phthisical; and a surviving girl is extremely scrofulous.

A fourth family (Hübchen) came in after the last mentioned, all healthy. The mother became phthisical, and bacilli were detected in the sputum. Two children died of meningitis under one year old.

Summing up the history, it appears that for eight years, 1865-73, the dwelling was free from tubercular disease. Then came one year's tenancy by a person already tuberculous. After this, in a period of twelve years, at least twelve cases of tubercular disease were traced to this source.

It is noted that the dwelling was never vacant, the new tenants entering while it was, so to speak, still warm from the last; and during the whole period it was never painted or cleaned.

Engelmann asserts that this neglect did not occur in other parts of the same large house; and that no similar instance of tubercular disease clinging to a dwelling was observed in them.*

The gigantic literature of tuberculosis, if sifted, would, no doubt, supply other similar instances; but as it is on an occasion like this impossible to be exhaustive, I will only assume that the evidence adduced proves that the tubercular infection is sometimes found to attach itself to houses; and that in some cases this infection is clearly traceable, as in acute contagious diseases, to contamination by a previous occupant. Even in cases where this cannot be proved, there is nothing to contradict the hypothesis that the infection may have been originally introduced by a tubercular individual. If, on the other hand, we should have instances of what is presumably tubercular house-infection, occurring in *new houses*, then, however unhealthy the situation, it would tell against this hypothesis, and would rather point to a miasmatic or ectogenic habitat of the bacillus.

It is clear that the hypothesis of house-infection is quite adequate also to explain cases attributed to direct personal contagion; since a present and living tubercular patient must contaminate a house or room even more certainly that one dead or departed.

As an instance of such cases I quote the following (*Collective Investigation Record*, 1883):—

"Miss R., aged 48, a dressmaker, living in a rather lonely cottage in Bedfordshire, had three apprentices, young girls of from 17 to 19 years of age, not related, from three adjacent villages, who took it in turn to remain in the house, and slept with her, each one for a week at a time. During their apprenticeship Miss R. was taken with phthisis, of which she died. In less than two years afterwards, all three apprentices died of phthisis, though no trace of this disease appeared in the family history of any of them; and the parents, brothers, and sisters of two of them are alive and well at the present day."

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* Since writing the above, I have come across another paper by Dr. Niven of Oldham (*Public Health*, vol iii, p. 463), containing notes of cases in which exposure to infection of tubercular disease was ascertained. From these I extract the following as bearing on house-infection, carefully omitting all cases of relationship:—

No. 17, man of 22. Previous tenant in the house where he contracted the disease died of consumption in 1886. House damp, unhealthy.

No. 34, child of 4 months, died in a house where previous occupants died of consumption, but child only lived five weeks in this house.

No. 57, man aged 24. After five years' illness, previous to which time he had lodged with a consumptive young man.

No 95. Lodged with a person who since became consumptive six years before. This period seems too long to account for the infection of either.

Dr. Niven states that his further researches into the occurrence of second deaths from tuberculosis in the same house do not yield so much information as might be expected. He thinks the facts suggest that the infection does not, as a rule, remain in a house sufficiently long to be dangerous to new occupants.

Dr. Niven, in an address reported in *Practitioner*, 1889, ii, 223, gives other cases where there was a probability of infection having been acquired in a particular house. The numbers were six out of forty-one houses, or about one-seventh. Still, in all these cases, as I understand, the element of family relationship also came into play.

I quote this case particularly since it has been criticised by Dr. Andrew, in his able Lumleian Lectures on the Causation of Phthisis (*Brit. Med. Journal*, 1884, i, p. 754), as inconclusive, because the three apprentices did not communicate the disease to their own families; and he regards it is unlikely that there should have been a special virulence in the first case which was wanting in the other three. The answer is, I think, that the house inhabited by Miss R. had become converted into a focus of contagion. The circumstances in the three families to which the apprentices belonged, we do not know; nor even whether they died of phthisis in their own families; but very probably the conditions necessary to make their houses foci of contagion were wanting.

Although I have purposely avoided quoting cases of multiple occurrence of phthisis in families, and especially the numerous instances of coincidences of the disease in husbands and wives, as instances of the effect of house-infection, it is clear that they can be accounted for on this theory with great facility; since, as a rule, the inhabitants of one house belong to one family. And even the supposition of a family predisposition to infection does not enfeeble the argument; since no infection spreads inevitably, and there must be some reason why one person is infected rather than another; while in contagious diseases like diphtheria and typhoid a family predisposition, irrespective of contagion, is often displayed. The hypothesis of direct inheritance, if clearly established, is the only one which makes all others unnecessary.

Another remarkable law which, on the principle of concomitant variations, tells in favour of house-infection, is the preponderance of phthisis in the female sex *among agricultural populations*, while the inverse proportion obtains in town. This fact is so well ascertained in our own country as to be beyond dispute. An excellent table is given in Dr. Ransome's Milroy Lectures (p. 39), showing twelve country districts with an excess of female phthisis, and nine town districts with an excess on the male side. The statistics of Meppen, formerly referred to, exhibit the same law. It is stated in Dr. Wilson Fox's work on *Diseases of the Lungs*, on the authority of Walshe, that, in certain malarious districts, the women who do not work in the fields are the only sufferers from phthisis.

The explanation generally given of this difference is that the outdoor life of men is healthier, and therefore antagonistic to phthisis. But it is as logical to say that the indoor life of women exposes them to some injurious influence derived from the house itself.*

Further, it may be observed that in the case of cattle the connection of tubercular disease with particular buildings, or even with particular stalls in a building, has often been observed, and is regarded as an established fact. Moreover, in the case of rabbits, or other rodents kept for experimental purposes, the production of tuberculosis in them by infection of the places in which they are kept is a well-recognised occurrence. This was the cause of the experimental fallacies which led to the belief that tubercle could be produced in animals by the inoculation of inflammatory non-specific material. Cohnheim found that he could produce tubercle by this kind of inoculation in rabbits in the Pathological Institute at Berlin, but when he removed to Würzburg these experiments no longer succeeded. The explanation was that the cages for rabbits, etc. in Berlin had become permanently infected with tubercle, and it has been recognised by Dr. Burdon Sanderson and the late Dr. Wilson Fox that the same explanation applied to their results.

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* The significance of this difference is not diminished, but enhanced, by the results of a valuable table on the relative liability of the two sexes to pulmonary phthisis, contributed to the same work (p. 519) by Dr. William Ogle. Dr. Ogle shows that, taking the country as a whole, this liability is the same for the two sexes when all question of age is put aside; but this equality does not extend to other tubercular diseases, under which headings the male death-rate is at all periods of life in excess of the female. This excess, too, is especially marked in the early years of life (when children of both sexes live equally in the house), the death-rate of male children from these diseases being for the first five years decidedly higher than that of females; the mean annual mortality from all tubercular diseases of one million children under five being, for males, 5,385, for females, 4,264, or an excess on the male side of more than 25 per cent. As regards phthisis alone, he finds that the male death-rate in the first year of life is more than twice as high as the female rate. In the second year it is still slightly higher, but in the third year the female death-rate begins to exceed the male, and continues to be in excess up to about 35 years of age, after which the male rate preponderates. This excess on the female side is especially marked in the period from ten to twenty years, when it amounts to 50 per cent. Now it is at this period of life that the difference between the outdoor life of boys and the indoor life of girls begins. The greater liability of the male sex to phthisis after 35 years of age seems, however, to be quite unexplained by such considerations.

Numerous direct observations have been made to the same effect; so that this is a danger well recognised in all such experimental researches, and guarded against with corresponding care. From the above evidence I think we may conclude that the tubercular infection is frequently found in houses, and that by residence in such houses human beings may become infected. Further, there is no reason to suppose that the bacilli, by which they become so, have had any other origin than from men, or possibly domestic animals, affected with the tubercular disease. How long such infection may persist we do not know.

Mode of Tubercular Infection from Houses.

The precise manner in which infection is acquired from dwellings has been much discussed, but the most definite results have been obtained by the researches of Dr. Cornet* and others who have followed his methods. To give a full account of his results would occupy too long. I can only glance at a few of the facts which he has collected.

His method consisted in removing small quantities of dust from the floor and walls of hospital wards, the private houses of phthisical patients, and other buildings; either with a platinum spatula, or by rubbing the wall with a sterilised sponge. The materials thus obtained were mixed with a small quantity of sterilized bouillon or distilled water, and injected into the peritoneal cavity of guinea-pigs, the utmost care being taken to avoid accidental infection, and to take animals which were free from disease; although a previously acquired tuberculosis is easily distinguished in these animals from an inoculated one. The method of inoculation is, I need hardly say, now recognised as a much more delicate test for the presence of tubercular infective material than microscopical examination for bacilli. In fact, in specimens of dust thus proved to be capable of transmitting tubercle, no bacilli could be detected, which, of course, does not by any means show that none were present, but that they were few in number, or else in the state of spores.

The part of the room chosen for investigation was one near the phthisical patient, but not one where sputum could have been directly deposited; and sometimes parts of the bedstead were also examined.

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* *Ueber Tuberkulose*, von Dr. Georg Cornet. Leipzig, 1890.

Some of Cornet's results are very instructive. First, as a negative conclusion, Cornet's researches give no support to the prevalent impression which we often hear expressed in an exaggerated form, that the tubercular virus is universally distributed in cities, in houses, or even in hospital wards. Out of seven hospitals investigated, five gave positive results, that is, a certain proportion of the animals inoculated with dust or other products became tuberculous. In some cases the animals died of some intercurrent disease, such as peritonitis or malignant œdema, showing that the dust contained infective material.

Specimens from three lunatic asylums, and from rooms where phthisical patients lived or had lately lived, gave positive results in a smaller proportion. Specimens from the dwellings of private consumptive patients gave positive results in nearly half the number of cases. What is very striking is that specimens from hospital out-patients' rooms, from a hospital for skin diseases where there were patients affected with lupus, from surgical wards, and from the Pathological Institute and an Orphan Asylum, all gave negative results as regards tubercular infection. As a further control experiment, specimens were taken from several public buildings, from the outer walls of houses in frequented streets, and from street dust, all of which gave no tubercular infection, though in many cases other diseases were produced.

The general result was to show that in many cases the places where phthisical patients contain dust which is capable of communicating tubercle by inoculation.

The negative results from other buildings and places could not, of course, prove that no tubercular material was there, but only that it was not universally distributed.

To a certain extent these results appear consoling, since it has been of late years so much the custom to assume that in cities, at all events, one is in continual danger of breathing in tubercle bacilli, and that we must all of us have many times had tubercle bacilli in our mouths or air-passages. Not, of course, that they exclude the possibility of infection by the most remote channels, but they indicate from what quarter infection is likely to come, if it does come.

Krüger has confirmed Cornet's results by investigations carried out at Bonn.

The chief objections to his views are drawn from the theoretical considerations that other modes of acquiring tubercular disease must exist, and are more important;

some authorities, as Baumgarten, trying to prove that tubercle is mainly inherited, and others that food in some form is the chief means of communicating the disease.

Is tubercular disease acquired by inhalation of bacilli?—Cornet's views on the spread of tuberculosis, as deduced from his experiments, lay an almost exclusive weight on infection of the lungs by inhalation of tubercle bacilli, which must be so dry as to be carried about with dust, and hence he regards moisture of walls and floors as a condition hindering, not favouring, the spread of infection. That tubercular sputum does contain the virus of tubercle in a very potent form is proved "up to the hilt" by countless inoculation experiments. It is not too much to say that the production of tuberculosis by this means in rodents is as certain as the communication of cow-pox by vaccine lymph.

Still the question is whether this is the chief means of spreading tubercular disease among mankind; and this resolves itself into two questions: first, whether the sputum is the chief means by which tubercle-infection leaves the body; and secondly, whether inhalation is the chief means by which it is received. The one does not quite necessarily follow from the other.

(1) As to the first question, it would seem that if the tubercle bacillus be regarded as a parasite, such a means of continuing its species is in accordance with the general laws of parasitism, and similar to those methods which animal parasites unconsciously adopt for the continuance of their species. Consider the case of a tapeworm continually expelling its eggs, amounting to many thousands in a year, from the intestines of its host; though only one here and there arrives at a destination where it is able to develop into a cysticercus; and even out of this number very few again are likely to undergo further development into a tapeworm.

Consider, then, the expulsion of tubercle bacilli from the lungs in numbers counted by millions (twenty millions in twenty-four hours, according to Bollinger), as compared to thousands in the other case; and suppose that only one here and there arrives in another body where it can develop: is not this *comparatis comparandis* a very similar case? Why, we may ask, should the tubercular parasite plant itself in this particular part of the body, and set up a chain of processes which, if regarded as intentional, would be a mechanism admirably constructed to facilitate the passage of the species from one host to another? It is self-evident that these processes are not intentional, in any sense in which that word can be used; but still, they may be the result of an inherited habit, impressed upon the bacillus in the course of its evolution through countless generations by which its existence as a parasitic species has become established. I have elsewhere* drawn attention to the fact that all the habits of animal parasites, the way in which they enter the body of their hosts, the part of the body to which they direct themselves, and the channel by which they leave it, all tend to the preservation of the race as a parasitic species, and, if contrived deliberately for that end, would be regarded as admirably adapted for the purpose. Thus the hydatid cyst is developed in the liver, the trichina spiralis passes into muscle; and thus both find their way, with food, into the bowels of their carnivorous host, where they undergo their next development.

Thus, too, the egg of the tapeworm finds its way into the water, with which it enters the body of that host in which it has to pass through its next stage of existence.

The law is, I believe, the same for vegetable parasites, such as the bacteria which constitute the contagia of specific diseases. They must enter the body by a suitable channel, and pass to a part from which they can again leave the body in such a way as to continue the race. Thus the air-borne contagia of scarlatina and small-pox are determined to the skin, from which they pass into the air again; the contagia of cholera and enteric fever pass out from the bowels into the soil and water; each passing into the medium best adapted to facilitate its passage into the next host, and thus secure the continuance of the race. In soil-diseases, such as ague, not communicable from man to man, we should not expect to find any elimination of the contagium or germ; and the *Plasmodium malariæ* of Laveran, which is not known to pass out of the body by any channel, certainly fulfils this negative condition.

The fact of the elimination of tubercular contagium on a large scale from diseased persons certainly, so far as it goes, points to the conclusion that tuberculosis is not a soil-disease (as suggested, for instance, by Dr. Andrew in his Lumleian Lectures), but belongs to the contagious family.

Another reason for thinking that the lung is the predominant, or one might say, figuratively, the appointed channel for the elimination of the tubercle bacillus, and consequent preservation of the parasitic race, is that by whatever means the bacillus enters the body, it ultimately, in a great majority of cases, affects the lungs. Louis's old law, that if tubercle is found anywhere in the body, it is found also in the lungs, still remains nearly true; the exceptions being chiefly in so-called scrofulous affections; though even these may produce general tuberculosis. Another still more cogent fact is that the experimental tuberculosis of animals, whether brought about by inoculation or by tubercular food, nearly always produces pulmonary tubercle as a secondary lesion; perhaps always, if the animals live long enough; and this even when the primary lesion is small or inconspicuous. This is attested by all experimenters, from Villemin onwards.

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* "Specific Diseases considered with reference to the Laws of Parasitism" *St. Thomas's Hospital Reports*, vol. xx.

(2) But the question whether inhalation of dried sputum into the lungs is *the chief means* by which tubercular disease is acquired is a very different one.

Against the assumption there are many weighty arguments, which have been forcibly urged by Baumgarten and others.

First, the artificial production of tubercular disease in animals by inhalation, though it has been actually effected, is a very difficult process.

Many experiments in this direction have completely failed, though Tappeiner, Klein, and others have obtained positive results. Tappeiner's were obtained by a special and difficult method. He shut up the experimental animals in a box or chamber, into which large quantities of tubercular sputum were discharged in the form of spray by a special apparatus, and found that they all became tuberculous. It may be said that such a concentration of virus does not occur in nature. An unfortunate occurrence showed, however, that infection of the human subject by this means was possible. The servant who had charge of Tappeiner's apparatus, in spite of the most forcible and serious warnings, persisted, by way of bravado, in entering the inhalation chamber. He was a very robust man, in perfect health, and without the slightest hereditary tendency to consumption. Nevertheless, he paid the penalty of his rashness by acquiring an acute tuberculosis, which carried him off in fourteen weeks, producing typical morbid appearances in the body.*

One positive fact outweighs many negative, and this would be enough to show the possibility of infection by inhalation; though such conditions may be hardly likely to occur in ordinary life. The inhalation of tubercular material sufficient to produce disease must, it may be conceded, be somewhat occasional, and governed by circumstances which can only be called accidental. Nevertheless, accidents will happen. Some may think, with Cornet, that inhalation accounts for the greater part of tubercular diseases. Some may think, with Baumgarten, that it explains only a small part. It seems impossible to deny that it explains a certain part. Supposing, however, it be proved that the sputum of phthisical persons sets free a large quantity of virulent material, is there no other way than inhalation by which this material can enter the human body?

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* Cornet, *Tuberkulose*, p. 117; from *Schweninger gesammelte Arbeiten*, Berlin, 1886, Bd. i.

There is certainly one other, that is, with food into the digestive channels. This must be a possible means, as seen in the rare cases of infection with typhoid or cholera of those who attend in sick-rooms, and who cannot be supposed to inhale the organisms of these diseases. It must be a rare case with adults; but for children seems not so impossible. Considering the habits of children, one can see how they might, in the crowded dwellings of the poor, pollute their articles of food with tubercular material scattered about the room of a phthisical parent, or other inmate. Among the causes of intestinal tubercular disease, so frequent in children of phthisical families among the working classes, this possibility should not, it seems to me, be forgotten.

There is, again, the possibility of inoculation; a well-known, though rare, means of acquiring the disease from a previous case of tuberculosis. This undoubtedly happens sometimes, but can hardly be a main factor in the continued existence of the bacillus.

Putting all these possibilities together, it may still seem that the probability of contagion, direct or indirect, is small compared with the probability which there is in the case of the diseases commonly called contagious. Moreover, the very word contagious calls up a group of relations which we cannot trace in tubercular disease, and which prejudice us against the reception of the idea. We think at once of scarlatina, measles, small-pox, and so on, in which the contagion may be briefly characterised as direct, easily traceable, and momentary; while the communication of tubercle, if it occurs, will be indirect, untraceable, and possibly requiring time for its completion.

But there is one consideration lost sight of in contrasting the communicability of tubercle with that of the acute specific diseases, and that is the element of *time*.

The period of contagiosity in acute disease is measured by days or weeks, that of tubercular disease by years. A patient with measles or small-pox emits an extremely virulent contagium during, say, three to six weeks, and the possibilities of transmitting the disease are in the main limited to that period of time.

On the other hand, a patient with phthisis goes on expectorating a much less virulent contagium, with fewer possibilities of transmission; but the process goes on for two or three years. Is it not possible that the longer duration of contagiosity may make up for its lower degree? The final result, as regards possibility of transmission, might be, if not equal, at all events sufficiently nearly so to be comparable. The total contagiosity, that is, the possibility of existence or *viability* of the specific micro-organism, is a function of two variables, duration and intensity; so that a high value for one of these variables may make up for a low value of the other. If the acute specific fevers had only a low degree of contagiosity they would probably cease to exist, so far as man is concerned, or, at least, cease to be transmitted (though their living causes might have another mode of existence).

But if tubercular disease had the degree of contagiosity seen in the acute specifics, the results, as regards its diffusion in the world, would be very different from what they are, and much more disastrous.

Hence a comparatively low degree of contagiosity for tubercle might still suffice to explain its continued existence, more especially if reinforced by other factors which I will now proceed to consider.

Hereditary Transmission of Tubercle.

There is another way, quite different from infection, in which a specific disease might be kept alive and remain endemic in any country or population: that is, by inheritance.

Let it once be granted that tubercular disease may be transmitted directly from parent to offspring, and a considerable proportion of the cases of disease might be explained.

The heredity of tubercular disease is a vast problem which can only be briefly touched upon here; but the broad fact that the children of tuberculous parents are, on the whole, more likely than others to become tuberculous, has never been doubted, at least in modern times. Two explanations may, however, be given of this fact. The one, which has been till lately the only accepted theory, is that what is inherited is a particular predisposition or tendency, by which members of tubercular families are more likely than others to acquire tubercular disease when exposed to any of its causes.

But since the discovery of the specific nature of tubercle, another theory, that of *direct inheritance*, has been proposed, of which Cohnheim was, I believe, the originator; and Baumgarten is now the chief supporter. It supposes that the germ of disease (*i.e.*, the bacillus) is directly transmitted either from the male or female parent, just as we know the germ of syphilis is transmitted, even though the precise nature of the latter is unknown.

In the case of syphilis the proof of such transmission rests upon the occasional presence of signs of the disease in the newly-born child, or the immature fœtus; and also on the occurrence at later periods of manifestations of syphilis belonging to the secondary or tertiary stage, even when strictly congenital symptoms were not observed, or perhaps did not exist, and contagion was out of the question. If the case of tubercle were parallel, we ought to be able to find occasionally evidence of fœtal or strictly congenital tuberculosis, and more frequently evidence of the development of the disease in early or adult life in circumstances under which direct acquisition of the disease was, to say the least, improbable.

CONGENITAL OR FŒTAL TUBERCULOSIS.

A strong argument for the possibility of fœtal tuberculosis in man is derived from the fact that it undoubtedly occurs in cattle.

There are now a considerable number of well-established cases where fœtal or newly-born calves have been found presenting characteristic lesions of tubercle, and in some of them the occurrence of tubercle in the mother was well established. In a smaller number of instances, I think, on the whole, tubercular disease was found to be present in the sire.

This is the more remarkable, as tubercle is very rare in calves at the age at which they are generally killed for the market: the development of the disease not usually occurring in cattle till the second year, or later.

A very small number of similar instances have been observed in human infants.

A definite observation of tubercle in a newly-born child was reported by Hiller to the Medical Congress at Copenhagen in 1884. Demme found tubercle of the thymus in a child dying at six weeks old, which, from the inaccessibility of that organ, could hardly have been acquired.*

A well-marked case of tubercle in the tissues of a fœtus removed by Cæsarean section from the body of a mother who shortly afterwards died from acute tuberculosis is recorded by Birch-Hirschfeld. Tubercle bacilli were detected in the fœtal liver, and the presence of the virus was proved in other organs by inoculations into animals. The bacilli were also found in both the maternal and fœtal portions of the placenta, showing that they were capable of passing through that organ.†

In the discussion on Birch-Hirschfeld's case, Rindfleisch reported a quite similar one.‡

In the last two cases the method of transmission was apparently through the placenta, which is not the same thing as deposition of bacilli in the ovum.

It has also been shown that the infective virus of tubercle may be contained in the tissues of the newly-born infant of a phthisical mother, even when the infant shows no visible tubercle (Landouzy and Martin), though it must be admitted that many experiments in this direction have failed. Landouzy has reported another case of the same kind in the five-months-old (abortive) fœtus of a phthisical mother. Transmission of the disease from the male parent direct to the ovum is also probable, though, from the nature of the case, less easily demonstrated. Tubercle bacilli have been found in the testicles or seminal vesicles of men dying from phthisis, even when those organs were free from disease (Jani); and such facts may also explain the occasional transmission of tubercular disease from husband to wife. Experiments to test by inoculation the infective power of the semen in similar cases in the human subject have in several cases given only negative results: but the semen of tubercular guinea-pigs was found by Landouzy to be infective in six cases out of sixteen.

Another line of evidence is derived from the presence of tubercle in very young children, in whom infection from without was, at all events, very improbable, if not absolutely excluded. There is, or was till lately, an impression among pathologists that tubercular disease is very rare in infants during the first year of life. But this may depend partly on the fact that children's hospitals do not admit many children so young, and that, consequently, few *post-mortem* examinations are made of infants dying at this age. The earliest instance in my own experience was that of a child who suffered (as was alleged, from the time of birth) from a scrofulous abscess, which ended at the age of four months in general tuberculosis. But in my experience such cases are not common, and their rarity is borne out by the results of the *post-mortem* examination of a number of still-born children made by my friend Dr. Cory. In one hundred bodies thus examined he never found any visible tubercular disease, though signs of congenital syphilis were sometimes present.* Dr. Cory informs me that a second series of one hundred cases has given equally negative results as regards tubercular disease. Considering that these statistics were collected in the Maternity Department of St. Thomas's Hospital in the south of London, from a population among whom tubercular disease is certainly widely prevalent, they are undoubtedly of some weight as showing that strictly congenital tubercular disease is far from common, and can hardly be an important factor in keeping alive the disease as an endemic.

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* For reference to a few other cases of the kind, see Dr. J. E. Squire's elaborate paper in our *Transactions* for 1888-9 (vol. viii, p. 51).

† *Wiener Mediz. Blätter*, No. 17, 1891.

‡ Baumgarten's *Jahresbericht*, 1890, p. 299.

Moreover, it will appear on reflection that tubercle developing thus early in life will have a very small chance of being transmitted to the next generation. For the infants thus affected will mostly die long before the age of reproduction; or, if they survive, it can only be on condition of the disease being completely cured, so as to be, we may presume, incapable of transmission. Infantile tuberculosis, if fatal to the subjects, will also be fatal to the bacilli producing it. Also, the chance of spreading the disease by infection will be much less in the case of infants than in that of adults. Young children do not expectorate, and if they did eliminate the bacilli in any numbers, their relations to society are so much more limited than those of adults, that the probabilities of infection are fewer. On the whole, then, from the point of view of the tubercle bacillus, the production of infantile or congenital tuberculosis is not a hopeful direction in which to propagate its race. Bacilli which had this habit of early development would die out, and the continuance of the species bacillus would be arrested so far as that particular strain was concerned.

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* Cory, *St. Thomas's Hospital Reports*, vol. viii, p. 61, and vol. x, p. 263.

Latency of the Tubercle Bacillus.

Baumgarten's theory of hereditary transmission of the bacillus requires another condition in its natural history. It assumes that the bacillus may remain latent in the human body, in the form of spores or some inactive state, till the period of adolescence or early adult life. He supposes that the growing tissues may exert an inhibitory effect on the growth of the bacillus, which will therefore only begin to develop when the period of growth is past. If it become active at this period of human life, it will have a chance of being further transmitted by inheritance (since tubercular individuals are by no means deficient in fertility or averse to matrimony); and will also have many more chances of being transmitted by indirect contagion (by expectoration and otherwise) than in the case of an infantile disease.

It would certainly seem, *à priori,* that the best chances for inherited bacilli, of transmitting their race, would be given, if they remained latent in the human body till adult life. The question is, whether we have any reason to believe that the bacilli may have this property or habit of latency.

It may seem at first sight very improbable that this should be the case. Nevertheless, such a fact would not be without analogy. The disease of silkworms called *pébrine,* has been shown to be caused by a micro-organism, which may be transmitted by contagion, and also by inheritance, to the eggs. In the latter case the eggs may be hatched, and the worm go through several changes, but its development is gradually arrested, and it dies, when its body is found to be filled with the microbes. On Baumgarten's theory, tubercle is a sort of *pébrine* of the human race; the inherited germ becoming incorporated with the ovum, but not destroying it, and not producing obvious disease till a more or less advanced stage of development is reached; while, like the silkworm disease, it has also another mode of transmission by external contagion.

In either disease, if the parasitic organism were fatal to its host at too early a period of development, hereditary transmission through more than one generation could not occur.

Furthermore, if we suppose that this late development or prolonged latency were a sort of *habit* of the parasitic micro-organism, we may see, on the principle of natural selection, that the habit might become permanently fixed in the bacillus. For those which developed late would have a much better chance of perpetuating their race than those which developed early; the latter, indeed, soon dying out. Thus the property of prolonged latency might become a permanent feature in the biology of the bacillus.*

In cattle, among which the continuance of tuberculosis, especially as regards inheritance, seems to be more certain and uniform than in the human race, the tendency to late development of this disease is most marked. This is shown in the first place by the great rarity of tubercle in calves slaughtered for food, as compared with its frequency in adult cattle; and is supported also, though less conclusively, by its greater frequency in cows than in bullocks, as observed in slaughter-houses. Bullocks being bred for the purposes of food are seldom allowed to live beyond the third year; while cows are generally killed for food after they have served for reproduction, and for giving milk: consequently their average age at death is much higher. The circumstance which weakens the force of this argument is that cows, being kept in stalls, live in less healthy conditions than young bullocks, which spend a great part of their life in open pastures. On the other hand, a good many bullocks are fed for the market in stalls, in conditions favourable to the development of tubercle. But to what proportion of beasts this applies, or how much of their life is spent in these conditions, I am quite unable to say. Bulls kept for breeding, also, of course, live longer than oxen killed for the market; and it is asserted that tuberculosis is very common among the former class of animals; but I am not acquainted with any statistics showing its relative or absolute frequency.

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*The effect of heredity, combined with late development, in causing the spread of a disease, is indeed, as it seems, actually traceable in the case of cancer. In the *Report of the Registrar-General for* 1889, Dr. Ogle points out that the mortality attributed to cancer is constantly increasing, and that, assuming the disease to be hereditary, as is generally, though not universally, accepted; and if, as undoubtedly is the case, "its development does not, as a rule, manifest itself till after the usual age of marriage and reproduction, it must follow, as a necessary arithmetical consequence, that the tendency shall spread wider and wider among the population." By arguments similar to those here used, it is shown that this will not apply to tubercular phthisis. (*Report*, p. xiii.)

If, however, the laws of inherited tubercle are such as have been here suggested, tuberculosis would be analogous to cancer in this respect.

Objections to the Theory of Latency.

Many objections have been made to Baumgarten's theory of the latency of inherited tuberculosis. For one thing, it may be said that the presence of tubercle bacilli, or their spores, in the ovum, must inevitably check its development and prevent it from arriving at maturity, if impregnated. Whether this would or would not be the case, is difficult to say, on *à priori* grounds.

As an analogy, we may recall the facts of inherited syphilis. When the syphilitic virus derived from either parent affects the impregnated ovum, it does not necessarily destroy its vitality. Abortion may occur, but in most cases the fœtus is completely formed, and, when born, generally shows at first no sign of the disease. But after birth the virus begins to develop, so that at a period after birth corresponding to the known incubation of the disease when acquired, the first signs of the disease, such as eruptions on the skin, etc., appear. It would seem as if the fœtal tissues exercised an inhibitory effect on the development of the syphilitic virus.

There are also some remarkable experiments bearing upon the effects of bacillary infection of an ovum, namely, those of Maffucci on hens' eggs. He introduced into impregnated eggs certain species of bacteria, choosing those which were known to affect the adult fowl. He found that these, especially pathogenic forms, were unable to grow in the tissues of the embryo, though they did not interfere with its development, becoming incorporated with it. The bacilli were, however, still capable of growth, so that at a later period, either during incubation, or when the eggs were hatched, they began to multiply, and produced a characteristic brood. It seemed as if the embryonic tissues, without killing the bacilli, exerted an inhibitory effect upon their growth. These results were afterwards extended to the tubercle bacillus; for when bacilli derived from tuberculosis of fowls were introduced into the impregnated egg, they remained latent, without injuring the embryo; but the young chickens, when hatched, became, in many cases, after the usual period of incubation, affected with typical tuberculosis.*

If this, or anything like it, should happen when the tubercle bacillus is enclosed in an impregnated human ovum, there might certainly be a hereditary tuberculosis developing after birth. But a period of latency extending till adult life, during which time the tissues inhibit the development of the bacillus, is longer both absolutely and relatively than in the case of the tubercular hen's egg.

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* For an account of Maffucci's researches, see Baumgarten's *Jahresbericht über pathogene Mikroorganismen*, 1887, p. 385; 1888. p. 180; 1889, p. 288.

Another objection to Baumgarten's theory of the transmission of latent tubercular disease is that the supposed heredity of tubercle is not always direct from parent to offspring. A sort of consanguineous tendency, that is, the occurrence of the disease in uncles, aunts, or grandparents, or even the mere fact of its occurrence in collateral relatives, being generally held to show a hereditary diathesis even when the parents were free from the disease. In all these cases (which are more numerous than those of direct parental transmission) we must go back to the grandparents to find the direct line of inheritance. Baumgarten supposes that the period of latency is here extended over two generations, that is to say, a tubercular parent transmits latent bacilli or their spores to his *grandchildren* passing through the body of the *children*, who are unaffected. This is to make a large demand upon the scientific imagination. We must suppose, for instance, that the female child of a phthisical parent may be born with bacilli or their spores lodged in the ova of her immature ovaries, though, perhaps, in no other part of her body; and these bacilli only develop when the ovum is impregnated, and born as a separate individual. The chief evidence to support this hypothesis is the alleged occurrence in the bodies of the children of tuberculous parents, for instance, in lymphatic glands, or other situations, of small masses of latent tubercle which may be discovered *post mortem*, never having undergone further development during life. With respect to this we must say that very minute and critical sifting of the facts is required.

I have presented this doctrine of direct hereditary transmission of the tubercle bacillus, as it is stated by its supporters, because, though by no means proved, it seems to be the only alternative or supplement to the theory of contagion, in explaining the continued existence of the bacillus as a parasite, and the consequent endemic continuance of tubercular disease.

It certainly seems to explain better than any other hypothesis the rare occurrence of fœtal tuberculosis, and possibly a good deal of the tubercular disease in early life. But to regard it, with Baumgarten, as the main factor accounting for tuberculosis in adults, seems to me an exaggerated way of looking at the question. Other facts which make against this explanation are the liability to phthisis of foreign races of mankind (*e.g.*, negroes) and wild animals kept in confinement, when they are brought into a tuberculous population such as that of England.*

Tuberculosis from Domestic Animals.

There still remains one possible factor in the continuance and preservation of tubercular disease in human societies, that is, its communication from domestic animals, especially from horned cattle. Great importance has been attached to this relation. It has been supposed that tubercle was originally a bovine disease which has been transmitted to man, and that its existence in our domestic breeds of cattle is responsible for a good deal of human tuberculosis. About its prevalence in cattle there can be no doubt; nor that it is propagated by the two means of contagion and inheritance; so that the bacillus is quite capable of continuing its species in the bovine race without living in any other medium, animated or inorganic. These facts have been clearly established by the observations of skilled veterinarians. The question for us is whether the bovine disease is transmitted to man.

If transmitted, it can hardly be in any other way than by the use as food of products derived from these animals, that is, of meat or milk. There can be little doubt that transmission of tubercle by these means is at least possible, and there is some evidence as to its probability, but the problem is a very complicated one. I do not propose to discuss it now, since the question is still under the consideration of the Royal Commission on Tuberculosis, of which I am a member; and it would be premature to express any individual opinion upon it. But no one can suppose that this is the principal means by which the disease is kept alive among men. If we had no milch cows, and ate no beef, we should still have tuberculosis; as may be seen by the example of countries like Japan, where milk is hardly used as an article of diet, and beef very little, while tubercular disease is far from uncommon. It cannot therefore be at most more than a contributory cause.

One curious question as regards animal tuberculosis is this: If domestic animals give us tubercle, is it not also possible that we may convey it to them? Is not a phthisical cowman or dairymaid a source of danger to the animals under his or her care, especially when they are shut up in stalls?

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* For Baumgarten's views see his *Lehrbuch der pathologischen Mykologie*, vol. ii, p. 626 *et seq.*

The same possibility applies in an eminent degree to another class of animals, namely, to wild animals kept in menageries. There is no reason, I think, to believe that in their native haunts they suffer from tuberculosis, yet we find that a large number of the animals dying in the Zoological Gardens die of consumption. It cannot be hereditary in their case (and this might be taken into account in weighing the influence of heredity in producing tubercle), nor can we believe that clay soil, dampness, confinement, and so forth, are more than contributory causes. The infection must come from some source in the country which these creatures have unwillingly adopted as their own. Is it our phthisical population, or is it an infection of their cages, which conveys to them the fatal bacilli? This question might be, in the interests of zoology, well worth consideration.

CONCLUSION.

Considering, finally, what is it that constitutes tuberculosis an endemic disease, and maintains its existence, I conclude that this is due to the continued existence in the country of a specific organism, the tubercle bacillus.

That this organism has no proper home in external nature. It remains to be proved that it ever lives, develops, and multiplies outside the body of some animals. So far as our present evidence goes, it is a true parasite, and carries on its parasitic existence predominantly in men and bovines.

Hence tuberculosis is not a soil disease; its existence and diffusion are connected with human societies; still, it may keep alive in human dwellings for a time as yet undetermined.

The influence of external circumstances on the prevalence of the disease is undoubted, and this question is of the highest practical importance; but such circumstances are not the determining cause of the disease.

The continuance of the species bacillus tuberculosis as a human parasite is secured by its transmission in two ways: by passage from one host to another, that is, by contagion direct or indirect; and by inheritance. The former of these methods is probably by far the most important, transmission by heredity being subordinate. Nevertheless, opinions still differ, and will perhaps always differ, as to the relative importance of these two factors.

The parasitism of the bacillus in bovine animals is quite independent of its human parasitism, but may contribute to the latter. The actual importance, however, of this factor in keeping up human tubercular disease is probably small.

The question, What conditions of weakness or vigour favour or resist the invasion of the human organism by the parasite? is an entirely different one, though its importance cannot be denied. Any conclusions which may be arrived at as to the immunity or predisposition of particular races or individuals will not affect the laws governing the distribution and transmission of the parasitic micro-organism.

In conclusion, gentlemen, I hope that I have shown sufficient reasons for regarding tuberculosis as a specific endemic disease, and therefore belonging to that class of diseases with which the Epidemiological Society has always concerned itself.